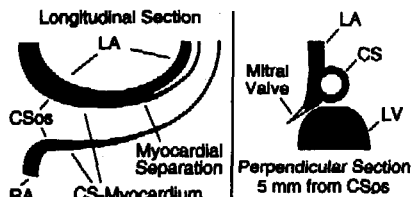


appeared continuous with RA myocardium (anterior and posterior to the CS Os) and the LA myocardium. The separation between the CS myocardium and LA myocardium was found at 30, 20 and 20 mm from the CS Os in the 3 hearts with longitudinal sections and at 30, 30 and 20 mm from the CS Os in the 3 hearts with perpendicular sections.



Conclusion: This canine study demonstrated RA to LA myocardial continuity via the CS myocardium. This may be an electrical RA-LA bridge which may be participating in atrial fibrillation and/or AVNRT.

9:00

775-3 Spontaneous conversion of atrial fibrillation to flutter in man: A stereotypical pattern of organization

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Atrial fibrillation (AF) often precedes typical atrial flutter (AFL), but the mechanism of this transition, in man, is unknown. We analyzed the mode of organization from multisite endocardial recordings in pts undergoing electrophysiologic evaluation, only analyzing episodes prior to ablation. Of 89 pts, 10 (3F, age 66 ± 6) had 17 episodes of AF which organized into typical AFL. Mean cycle length (CL) was calculated using electronic calipers for: a) 25 cycles starting 5 s after onset of AF; b) just prior to a more organized pattern of AF; and c) immediately prior to conversion to AFL. Activation sequence was analyzed using a 20-pole catheter for mapping the right atrium (RA). **Results:** The onset of AF was spontaneous (2), due to rapid pacing (9) or internal cardioversion (6). AF duration was 169 ± 229 s (range 15–862). Prior to conversion to AFL, a characteristic sequence of events was present in all 17 episodes. First, a gradual increase in AF CL was found (from 146 ± 26 ms after onset to 165 ± 28 ms; $p < 0.01$). Then, a sudden change in activation sequence was noted such that the lateral RA free wall was not activated for 264 ± 44 ms, and RA activation organized into a nearly identical sequence to that which would eventually be present during AFL [counterclockwise ($n = 14$); clockwise ($n = 3$)], though the rhythm was still AF (CL = 179 ± 23 ms). This more organized AF lasted from 2 to 49 beats. Finally, with another sudden change in CL (maximum delay on RA free wall = 277 ± 51 ms), typical AFL (CL = 242 ± 39 ms) was established. **Conclusion:** We speculate that three discrete events presage the conversion from AF to AFL: 1) Slowing of AF; 2) coalescence of multiple wavelets into a single unstable rapid wave in the RA; 3) extension of a line of block which stabilizes typical AFL to a longer cycle length. Higher density recordings will be needed to test this hypothesis. Nevertheless, a remarkably stereotypical pattern of endocardial organization is present during the conversion from AF to AFL in man, and the RA free wall sequence during organized AF predicts the direction of rotation of established AFL.

9:15

775-4 Evidence for involvement of the atrial insertion of accessory pathways in the genesis of atrial fibrillation

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Patients (pt) with accessory atrioventricular pathways (AP) present a higher incidence of paroxysmal atrial fibrillation (AF) than pt without accessory atrioventricular connections. Successful ablation of AP reduces AF significantly but recurrence of AF still exceeds that of a matched control population. The underlying mechanism remains unclear. We hypothesized that not only retrograde AP conduction but also reentrant circuits in the branching of the atrial insertion of the AP can trigger AF. We investigated the recurrence rate of AF after ablation of either the atrial or the ventricular insertion of AP. **Results:** 38pt with AP and paroxysmal AF without structural heart disease were included. 22pt had bidirectional, 14pt retrograde, and 2pt antegrade conducting AP. In 20pt AP was ablated at the ventricular insertion (group I), in 18pt at the atrial insertion (group II). Early recurrence of AP required a second session in 3pt. Mean follow-up was 26 ± 12 months and included Holter monitoring, questionnaire and electrophysiological re-evaluation in case of tachyarrhythmic events. 6pt (15.8%) experienced recurrence of sustained tachyarrhythmic

palpitations (group I: 5pt, group II: 1pt, $p < 0.05$). AF could be documented in 4 pt. Re-evaluation demonstrated in all cases persistent ablation success. 2pt had abnormal intraatrial conduction patterns (group I: 1pt, group II: 1pt). In all remaining 4pt AP was ablated at the ventricular insertion (group I). None of the pt with inobstructive intraatrial conduction and atrial ablation site experienced recurrence of AF ($p < 0.05$). **Conclusion:** Pt in whom AP was ablated at the ventricular insertion have more often recurrence of AF than pt with an atrial ablation approach. This finding gives evidence that not only retrograde AP conduction but also reentrant circuits in the branching of the atrial insertion of AP can trigger AF.

9:30

775-5 Anatomic determinants of atrial fibrillation cycle length in canines with chronic atrial fibrillation

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Atrial fibrillation cycle length (AFCL) is a function of tissue refractoriness, but the influence of anatomic structures on local AFCL is unknown. **Methods:** Chronic AF was induced in 13 dogs by creating mitral regurgitation and rapidly pacing the right atrium (RA) for ≥ 8 weeks. Electrograms (32 bipoles) were recorded in each atrium via a basket electrode catheter (EPT). The AFCL at each bipole was calculated as the mean of 25 consecutive interelectrogram intervals. Comparisons were made between 1) the anterior versus posterior regions of the RA, 2) the anterior versus posterior regions of the left atrium (LA), and 3) perivalvular bipoles in close proximity (≤ 1 cm) to the AV valve annulus versus non-perivalvular (> 1 cm from annulus) in the same region. The temporal stability of AFCL was assessed by correlating AFCLs obtained during two time intervals. **Results:** A linear correlation ($r = 0.86$, $p < 0.001$) was present between AFCLs obtained during different time intervals. The mean AFCL for the anterior and posterior RA and LA, and the perivalvular and non-perivalvular regions are displayed in the table.

RA Anterior	116 ± 20 ms	RA Posterior	112 ± 21 ms	$p = 0.38$
LA Anterior	104 ± 17 ms	LA Posterior	94 ± 15 ms	$p = 0.01$
Perivalvular	126 ± 34 ms	Non-perivalvular	104 ± 21 ms	$p = 0.05$

Conclusions: AFCL is stable over time. The posterior LA demonstrates a shorter AFCL than other regions. This area may be important for AF propagation. The AV valves are anatomic barriers to conduction which may block AF reentrant wavelets thus prolonging perivalvular AFCL.

776 Ischemic Preconditioning Limits Injury: What Are the Relevant Mechanisms?

Wednesday, March 19, 1997, 8:30 a.m.–10:00 a.m.
Anaheim Convention Center, Room A10

8:30

776-1 Reductions in Myocardial Oxygen Consumption Following Repeated Brief Coronary Occlusions in Chronically Instrumented Dogs — A Possible Contributing Mechanism in Preconditioning

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The protective effect of a preconditioning stimulus is believed ultimately to involve a reduction in energy demand and metabolism during subsequent prolonged ischemia. The possibility that a preconditioning stimulus down-regulates myocardial O_2 consumption (MVO_2) prior to sustained ischemia has been thought unlikely, i.e., reduced contractile function following a preconditioning stimulus has been assumed to represent "stunned" myocardium accompanied by a paradoxically high level of O_2 demand. To test this possibility directly, we measured regional MVO_2 and subendocardial segment shortening (SS) following repeated brief 2-min coronary occlusions in six chronically instrumented dogs. Following as few as five occlusions, SS and MVO_2 decreased systematically, averaging $79 \pm 5.1\%$ [SEM] and $83 \pm 1.6\%$ of control values (both $p < 0.05$ ANOVA). The reductions persisted with additional occlusions; following a total of 15 cycles, SS and MVO_2 averaged $81 \pm 5.5\%$ and $78 \pm 3.2\%$ of control, respectively (p again < 0.05 in both cases). Reductions in MVO_2 resulted predominantly from decreases in regional coronary flow, which averaged $84 \pm 2.8\%$ of control after five occlusions and $81 \pm 2.3\%$ after 15 occlusions ($p < 0.05$ in both cases). All parameters returned to pre-intervention values within three hours following the final occlusion. In five similarly instrumented "sham" animals, MVO_2 and